We Claim:

1. A method for treating a glycolipid storage-related disorder, comprising administering a therapeutically effective amount of an inhibitor of glycolipid synthesis in combination with an agent capable of increasing the rate of glycolipid degradation.

2. The method of claim 1, wherein the inhibitor of glucosylceramide synthesis is an imido sugar.

3. The method of claim 2, wherein the imido sugar is selected from the group consisting of N-butyldeoxynojirimycin (NB-DNJ), N-butyldeoxygalactonojirimycin (NB-DGN), and N-nonyldeoxynojirimycin (NN-DNJ).

4. The method of claim 3, wherein the imido sugar is N-butyldeoxygalactonojirimycin (NB-DGN)

5. The method of claim 1, wherein the inhibitor is selected from the group consisting of 1-phenyl-2-decanoylamino-3-morpholino-1-propanol (PDMP), D-threo-1-phenyl-2-decanoylamino-3-morpholino-1-propanol or a structurally related analogue thereof.

6. The method of claim 1, wherein the inhibitor is a nucleic acid encoding a peptide or protein capable of inhibiting glycolipid synthesis.

7. The method of claim 6, wherein the nucleic acid is an antisense sequence.

8. The method of claim 6, wherein the nucleic acid is a catalytic RNA capable of interfering with the expression of enzymes responsible for glycolipid synthesis.

9. The method of claim 1, wherein the inhibitor of glycolipid synthesis is an inhibitor of neuronal glycolipid synthesis.

10. The method of claim 1, wherein the agent capable of increasing the rate of glycolipid degradation is an enzyme involved in glycolipid degradation.

11. The method of claim 10, wherein the enzyme is selected from the group consisting of glucocerebrosidase, lysosomal hexoseaminidase, galactosidase, sialidase, and glucosylceramide glucosidase.

12. The method of claim 1, wherein the agent capable of increasing the rate of neuronal glycolipid degradation is a molecule which increases the activity of a glycolipid degrading enzyme.

13. The method of claim 1, wherein the agent capable of increasing the rate of neuronal glycolipid degradation is a nucleic acid sequence which encodes a neuronal glycolipid degrading enzyme.

14. The method of claim 1, wherein the glycolipid storage-related disorder is selected from the group consisting of Gaucher disease, Sandhoff's disease, Fabry's disease, Tay-Sach's disease, Niemann-Pick disease, GM1 gangliosidosis, Alzheimer's disease, stroke, and epilepsy.

15. The method of claim 1, wherein the inhibitor of glycolipid synthesis and the agent capable of increasing the rate of glycolipid degradation are given simultaneously, sequentially, or separately.

16. A method for treating a glycolipid storage-related disorder, comprising administering a therapeutically effective amount of an inhibitor of glycolipid synthesis in combination with bone marrow transplantation.

17. The method of claim 16, wherein the inhibitor of glucosylceramide synthesis is an imido sugar.

18 . The method of claim 17, wherein the imido sugar is selected from the group consisting of N-butyldeoxynojirimycin (NB-DNJ), N-butyldeoxygalactonojirimycin (NB-DGN), and N-nonyldeoxynojirimycin (NN-DNJ).

19. The method of claim 18, wherein the imido sugar is N-butyldeoxygalactonojirimycin (NB-DGN)

20. The method of claim 16, wherein the inhibitor is selected from the group consisting of 1-phenyl-2-decanoylamino-3-morpholino-1-propanol (PDMP), D-threo-1-phenyl-2-decanoylamino-3-morpholino-1-propanol or a structurally related analogue thereof.

21. The method of claim 16, wherein the inhibitor is a nucleic acid encoding a peptide or protein capable of inhibiting glycolipid synthesis.

22. The method of claim 21, wherein the nucleic acid is an antisense sequence.

23. The method of claim 21, wherein the nucleic acid is a catalytic RNA capable of interfering with the expression of enzymes responsible for glycolipid synthesis.

24. The method of claim 16, wherein the inhibitor of glycolipid synthesis is an inhibitor of neuronal glycolipid synthesis.

25. A pharmaceutical composition useful for the treatment of glycolipid storage-related disorders, comprising a therapeutically effective amount of an inhibitor of glycolipid synthesis, an agent capable of increasing the rate of glycolipid degradation, and a pharmaceutically acceptable carrier.

26. The pharmaceutical composition of claim 25, wherein the inhibitor of glucosylceramide synthesis is an imido sugar.

27. The pharmaceutical composition of claim 26, wherein the imido sugar is selected from the group consisting of N-butyldeoxynojirimycin (NB-DNJ), N-butyldeoxygalactonojirimycin (NB-DGN), and N-nonyldeoxynojirimycin (NN-DNJ).

28. The pharmaceutical composition of claim 27, wherein the imido sugar is N-butyldeoxygalactonojirimycin (NB-DGN)

29. The pharmaceutical composition of claim 25, wherein the inhibitor is selected from the group consisting of 1-phenyl-2-decanoylamino-3-morpholino-1-propanol (PDMP), D-threo-1-phenyl-2-decanoylamino-3-morpholino-1-propanol or a structurally related analogue thereof.

30. The pharmaceutical composition of claim 25, wherein the inhibitor is a nucleic acid encoding a peptide or protein capable of inhibiting glycolipid synthesis.

31. The pharmaceutical composition of claim 30, wherein the nucleic acid is an antisense sequence.

32. The pharmaceutical composition of claim 30, wherein the nucleic acid is a catalytic RNA capable of interfering with the expression of enzymes responsible for glycolipid synthesis.

33. The pharmaceutical composition of claim 25, wherein the inhibitor of glycolipid synthesis is an inhibitor of neuronal glycolipid synthesis.

34. The pharmaceutical composition of claim 25, wherein the agent capable of increasing the rate of glycolipid degradation is an enzyme involved in glycolipid degradation.

35. The pharmaceutical composition of claim 34, wherein the enzyme is selected from the group consisting of glucocerebrosidase, lysosomal hexoseaminidase, galactosidase, sialidase, and glucosylceramide glucosidase.

36. The pharmaceutical composition of claim 25, wherein the agent capable of increasing the rate of neuronal glycolipid degradation is a molecule which increases the activity of a glycolipid degrading enzyme.

37. The pharmaceutical composition of claim 25, wherein the agent capable of increasing the rate of neuronal glycolipid degradation is a nucleic acid sequence which encodes a neuronal glycolipid degrading enzyme.

38. The pharmaceutical composition of claim 25, wherein the glycolipid storage-related disorder is selected from the group consisting of Gaucher disease, Sandhoff's disease, Fabry's disease, Tay-Sach's disease, Niemann-Pick disease, GM1 gangliosidosis, Alzheimer's disease, stroke, and epilepsy.